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A METHOD OF SOFT BODY ARMOR EVALUATION: CARDIAC TESTING

by

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(U) The Law Enforcement Assistance Administration (LEAA) is sponsoring a program to develop an inconspicuous, lightweight body armor that will provide protection from standard handgun threats. A high-speed projectile that impacts but does not penetrate flexible armor material transmits large amounts of energy to the underlying tissues.

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20. ABSTRACT (Contd)

The goal of this study was to determine the hazards resulting from impacts with the .38-caliber, 158-grain bullet, at approximately 800 feet per second. The missiles impacted 7 plies of Kevlar 29 (400/2 denier) directly over the myocardium.

Extensive cardiac monitoring of 23 anesthetized Angora goats was performed. The study revealed that small myocardial contusions, damage to the valvular structures, and great vessel injury can be produced.

PREFACE

The work described in this report was supported by Project LEAA-J-IAA-005-4 awarded by the Law Enforcement Assistance Administration, US Department of Justice, under the Omnibus Crime Control and Safe Streets Act of 1968, as amended. This work was started in May 1974 and completed in February 1975. The experimental data are contained in notebooks MN 2549 and MN 1982.

In conducting the research described in this report, the investigators adhered to the "Guide for the Care and Use of Laboratory Animals" as promulgated by the Committee on Revision of the Guide for Laboratory Animals Facilities and Care of the Institute of Laboratory Animals Resources, National Research Council.

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A METHOD OF SOFT BODY ARMOR EVALUATION: CARDIAC TESTING

I. INTRODUCTION.

Kevlar 29, a proprietary product of the E. I. du Pont de Nemours and Co., is a synthetic fiber of the polyamine B type. This relatively new material has the highest ratio of tensile strength to weight of any man-made fiber. After extensive ballistic and animal testing, this material was selected for fabrication into inconspicuous lightweight bulletproof jackets.

Initial testing indicated that 7 plies of 1000-denier Kevlar 29 would prevent the penetration of a 158-grain, .38-caliber bullet with a striking velocity of 800 feet per second. This armor configuration prevented bullet penetration but the resultant armor deformation produced visceral damage in both the thoracic and abdominal regions.[1]

Because of the physiological importance and relative vulnerability of the heart, this study was undertaken to evaluate the trauma resulting from impacts directly over the heart.

II. MATERIALS AND METHODS.

Twenty-five Texas Angora goats were utilized for the experiment. These animals had identifying numbers 23050 through 23074. Animal 23068 was dropped from the study when necropsy revealed massive right lung pneumonia. Animal 23072 was dropped because of a malfunction in the missile-velocity-recording equipment. Henceforth, individual animals will be referred to by the last two digits of the identifying number.

Of the 23 remaining animals, 22 were impacted by a standard .38-caliber, 158-grain bullet, while wearing 7 plies of Kevlar 29. These animals weighed from 36.8 to 77.4 kg and the mean weight was 49.0 kg. The mean projectile velocity for these 22 shots was 823 feet per second (790 to 853 feet per second).

One additional animal (63) was impacted with a standard .45-caliber, 234-grain bullet at 813 feet per second. This animal weighed 73 kg.

An experimental protocol was initially formulated as follows: The animals were premedicated with 50 mg acepromazine via a percutaneously placed jugular catheter. This was followed by intravenously administered pentobarbital 65 mg/cc to produce general anesthesia (about 6 cc was required). The animals were then intubated and placed in a standing position in a stanchion. A 7-cm polyethylene catheter PE No. 320 was inserted percutaneously into the right common carotid artery to monitor blood pressure and to measure cardiac output. X-rays were taken to assure that the target point for ballistic impact was directly over the heart. This was usually in the fourth intercostal space on the left chest approximately 6 cm from the left sternal border, near the point of maximum cardiac impulse (PMI), as found by stethoscope.

The 23 animals tested can be divided into 3 groups according to modifications in the experimental protocol as testing progressed.

[1] Goldfarb, M. A., *et al.* EB-TR-74073. A Method of Soft Body Armor Evaluation. January 1975.

A. Group I.

In this group of seven animals, a PE No. 230 catheter was placed percutaneously via the left common carotid into the left ventricle to monitor end diastolic pressure. Correct positioning of the catheter was ascertained by recording typical ventricular pressure curves.

Prior to missile impact, electrocardiograms and measurements of systolic blood pressure, left-ventricular end-diastolic pressure, serum creatinine phosphokinase (CPK), serum lactic acid dehydrogenase (LDH), and cardiac output were obtained. Cardiac output was measured using the dye dilution method with indocyanine green using a Waters D400 densitometer and a Waters C04 analog computer.

After the armor was strapped to the animal, the animal was impacted with the .38-caliber, 158-grain projectile. The animal was shot during maximum expiration with electrocardiographic monitoring during the shot and for 1 minute after impact. The period of maximum expiration was employed to allow for "opening of the cardiac window" by the collapse and retraction of the cardiac portion of the left lung covering the heart. Projectile velocities were recorded for all shots.

At 30 minutes after impact, all parameters monitored prior to impact were recorded again. The animals were then extubated, allowed to recover from anesthesia, and given access to food and water.

At 24 hours after impact the animals were reanesthetized, intubated, and positioned in the stanchion. All parameters were again measured. The animals were then rapidly sacrificed with an intravenous bolus injection of pentobarbital. Complete autopsies were performed immediately.

B. Group II.

This group consisted of seven animals. The protocol as presented in group I was used except for the deletion of the left-ventricular end-diastolic pressure catheter and the addition of arterial (A) and venous (V) blood gas measurements for pO_2, pCO_2, and pH. Arterial and venous gases and pH were determined prior to the shot and at 15, 30, 45, and 60 minutes, and 24 hours after the shot.

C. Group III.

This group consisted of nine animals. The protocol used was the same as the protocol for group II with one exception: the projectile was fired during maximum diastolic filling of the heart and maximum expiration. This was accomplished by firing the projectile at end expiration between completion of the QRS wave and end of the T wave on the electrocardiogram.

III. RESULTS.

In earlier work at Edgewood with Texas Angora goats protected with 7-ply Kevlar armor, the .38-caliber 158-grain bullet was fired at the goat sternum (midsternal targeting). No morphological or electrophysiological cardiac damage was produced, primarily because the goat sternum is massive and succeeds in absorbing most of the projectile's impact energy.

Firing the projectile at the left chest of the animal affords one the opportunity to produce substantial amounts of damage to the goat heart. The chest wall thickness in the goat

measures approximately 3.0 to 3.5 centimeters and the heart is immediately adjacent to the parietal pleura of the left chest wall. Thus we believe the maximum morphologic and electrical cardiac damage are produced by an impact on the left chest over the PMI.

A. Group I.

Results for this group are summarized in table 1. In this group of seven animals, no complete penetration of the armor occurred. The velocities of the projectiles ranged from 790 to 845 ft/sec. All animals of this group experienced a small skin laceration secondary to the deformation of the body armor against the chest wall. This skin laceration measured approximately 2 cm extending down through latissimus dorsi and the serratus muscles to the underlying rib. No rib fractures or perforation of the pleural space occurred.

Of the seven animals, two (50 and 53) had small contusions of the cardiac lobe of the left lung, approximately 5 cc in volume. No morphologic cardiac damage occurred. The projectile velocities in these shots were 790 and 823 ft/sec.

One animal (52) had a 0.5-cm partial thickness contusion in the cardiac apex, associated with a small pulmonary contusion (left cardiac lobe). See figure 1.

One animal (54) had no cardiac damage, but this animal experienced injuries to both the aorta and right lung. The aorta exhibited a full-thickness transverse laceration in the ascending portion approximately 3 cm in length. There was a parietal pleural tamponade which prevented massive hemorrhage. A partial-thickness 2-cm transverse laceration of the aorta with intimal disruption also occurred approximately 3 cm from the aortic valve. Examination revealed multiple contusions involving the entire right lung (figures 2 and 3). The velocity of the projectile was 830 ft/sec.

One animal (55) had a small partial-thickness (0.1- by 0.2-cm) cardiac contusion along the anterior lateral segment of the left ventricle immediately below the left atrium, associated with a tear of the noncoronary cusp of the aortic valve. The projectile velocity in this case was 831 ft/sec.

One animal (56), struck with a .38-caliber projectile at 845 ft/sec, experienced multiple contusions involving the entire right lung. No cardiac or left lung damage was found.

Finally, one animal (51) impacted at 835 ft/sec had no damage.

1. Physiologic Monitoring.

a. Cardiac Output.

All animals experienced a depression of cardiac output at 30 minutes and 24 hours post shot. We observed a similar depression of cardiac output whether cardiac damage was present or absent.

b. EKG.

Nonspecific T wave changes were noted in most animals at 24 hours. No significant electrocardiographic abnormalities, conduction defects, or arrhythmias were noted immediately post shot, at 30 minutes or at 24 hours.

Table 1. Summary of Results for Group I

Animal number	Weight	Cardiac output[a]		Velocity	Projectile	Systolic BP		Left vent end diastolic pressure		Enzymes[b]	LDH	CPK	Pathology
	kg	ℓ/min		ft/sec		mmHg					units		
23050	42.2	Control	8.16	790	.38-caliber	Control	204	NA[c]		Control	620	5.2	Small left lung contusion
		30 min	7.52			30 min	188			30 min	780	7.6	
		24 hr	7.86			24 hr	192			24 hr	730	23.2	
23051	44.5	Control	7.20	835	.38-caliber	Control	160	Control	8	Control	630	3.5	No damage
		30 min	4.16			30 min	172	30 min	8	30 min	650	6.4	
		24 hr	4.04			24 hr	168	24 hr	8	24 hr	880	20.6	
23052	47.6	Control	6.96	834	.38-caliber	Control	144	Control	6	Control	700	24.1	Contusion heart
		30 min	3.80			30 min	128	30 min	4	30 min	640	42.4	
		24 hr	5.60			24 hr	92	24 hr	6	24 hr	850	68.8	
23053	46.6	Control	3.32	823	.38-caliber	Control	172	Control	8	Control	600	6.7	Contusion left lung
		30 min	3.0			30 min	160	30 min	36	30 min	800	15.6	
		24 hr	3.92			24 hr	160	24 hr	8	24 hr	1180	48.2	
23054	42.7	Control	3.12	830	.38-caliber	Control	152	Control	16	Control	NA	NA	Aortic lacerations
		30 min	3.28			30 min	140	30 min	4	30 min	NA	NA	Multiple right lung
		24 hr	2.72			24 hr	120	24 hr	8	24 hr	880	16	contusions
23055	36.8	Control	3.52	831	.38-caliber	Control	112	Control	8	Control	700	11.6	Torn aortic valve
		30 min	3.40			30 min	120	30 min	8	30 min	600	46.2	Cusp and small left
		24 hr	3.20			24 hr	128	24 hr	4	24 hr	1370	96	ventricular contusion
23056	56.2	Control	4.56	845	.38-caliber	Control	180	Control	12	Control	630	8.5	Multiple right lung
		30 min	3.24			30 min	192	30 min	12	30 min	720	22.0	contusion
		24 hr	3.84			24 hr	144	24 hr	24	24 hr	1040	26.4	

[a]Control values, goat: CPK, 0-12 sigma units; LDH, 400-600 units; 4.9 ± 1.5 ℓ/min (50 animals).

[b]Lactic acid dehydrogenase and creatinine phosphokinase.

[c]Not available.

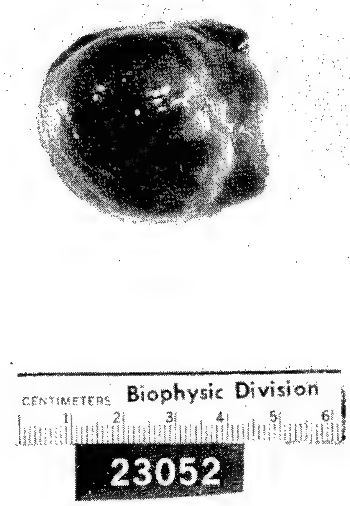

Figure 1. Partial Thickness Contusion (0.5 cm) in Cardiac Apex

Figure 2. Aortic Lacerations

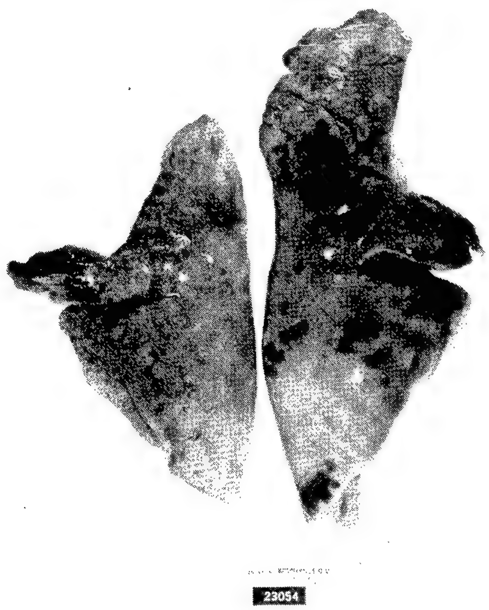

Figure 3. Multiple Pulmonary Contusions, Right Lung

c. Left Ventricular End Diastolic Pressure.

The left-ventricular end-diastolic pressure was elevated at 24 hours in one animal (56) with multiple right lung contusions. No other animal exhibited major changes in left-ventricular end-diastolic pressure.

d. Systolic Blood Pressure.

The systolic blood pressure was depressed in most animals at 24 hours (20% to 40%). No consistent relation between the level of damage and systolic blood pressure was observed.

e. Serum CPK and LDH.

The serum CPK and LDH rose in all animals 30 minutes post shot and at 24 hours. No consistent relation between the level of cardiac and pulmonary damage and serum enzymes measured was observed.

B. Group II.

This group consisted of seven test animals. The results are summarized in table 2.

Two animals (58 and 62) had small left lung contusions over the cardiac lobe. No cardiac or right lung damage was noted. The velocities of the projectiles used were 838 and 850 ft/sec.

Table 2. Summary of Results for Group II

Animal number	Weight	Cardiac output		Velocity	Projectile	Systolic BP		Enzymes			Arterial blood gas				Venous blood gas			Pathology
									LDH	CPK		pO_2	pCO_2	pH	pO_2	pCO_2	pH	
	kg	ℓ/min		ft/sec		mmHg			units			mmHg		units	mmHg		units	
23057	37.2	Control	4.08	834	.38-caliber	Control	132	Control	480	5.4	15 min	128.6	38.2	7.437	42.1	42.9	7.403	Aortic valve
											30 min	132.2	36.8	7.409	41.1	38.4	7.396	injury
		30 min	2.92			30 min	144	30 min	500	14.2	45 min	132	35.7	7.411	38	40.2	7.364	
											60 min	133	35	7.430	44.2	41.8	7.318	
		24 hr	3.28			24 hr	188	24 hr	1750	106	24 hr	89.6	33.3	7.455	35.8	36.2	7.406	
23058	52.8	Control	8.08	838	.38-caliber	Control	156	Control	800	6.1	15 min	110	42	7.457	62.4	42.7	7.437	Left lung
											30 min	105.2	42.6	7.435	62.2	42.1	7.422	contusion
		30 min	6.76			30 min	168	30 min	840	7.2	45 min	No sample			No sample			
											60 min	111.8	42.7	7.431	60.6	41.7	7.396	
		24 hr	4.96			24 hr	192	24 hr	520	8.8	24 hr	99	35.4	7.433	36.1	37.8	7.372	
23059	61.2	Control	5.04	840	.38-caliber	Control	188	Control	420	5.3	15 min	92.9	34.9	7.374	58.5	35.7	7.376	No damage
											30 min	105.8	28.7	7.421	55.3	31.8	7.377	
		30 min	5.40			30 min	160	30 min	450	11.2	45 min	93.4	31.4	7.407	42.5	32.9	7.380	
											60 min	105	31.8	7.420	48	32.7	7.391	
		24 hr	5.16			24 hr	188	24 hr	670	37.0	24 hr	94	28.7	7.410	48.1	30.8	7.395	
23060	52	Control	3.24	834	.38-caliber	Control	156	Control	990	10.9	Control	69.7	32.4	7.317	50.5	35.1	7.320	No damage
											15 min	70	31.1	7.353	52.3	32.3	7.339	
		30 min	2.80		.38-caliber	30 min	168	30 min	970	13.2	30 min	79.8	30.0	7.345	49.5	32.2	7.335	
											45 min	71.2	30.2	7.350	45.7	30.7	7.331	
		24 hr	3.92			24 hr	160	24 hr	950	101.0	60 min	65.0	29.6	7.368	41.5	30.6	7.340	
											24 hr	92.6	33.9	7.440	43.5	36.3	7.380	
23061	50.2	Control	6.60	852	.38-caliber	Control	152	Control	470	4.5	Control	102.9	31.7	7.375	73.8	37.4	7.366	No damage
											15 min	107.1	34.2	7.378	73.6	35.7	7.357	
		30 min	5.24			30 min	168	30 min	570	8.0	30 min	97.3	36.5	7.344	65.9	36.8	7.339	
											45 min	101.3	37.8	7.378	68.3	35.8	7.363	
											60 min	101.6	36	7.367	67.3	36.7	7.354	
		24 hr	6.16			24 hr	112	24 hr	NA	NA	24 hr	87.3	25.2	7.476	45.5	25.9	7.460	
23062	44.4	Control	6.64	850	.38-caliber	Control	172	Control	620	4.8	Control	67.8	36.4	7.348	44.9	38.7	7.291	Left lung
											15 min	54.6	36.4	7.349	43.9	36.4	7.305	contusion
		30 min	4.36			30 min	148	30 min	NA	NA	30 min	52.4	34.6	7.387	39.2	34.9	7.352	
											45 min	56.0	33.4	7.371	42.7	36.0	7.350	
											60 min	52.3	34.4	7.378	38.1	35.3	7.360	
		24 hr	4.52			24 hr	104	24 hr	NA	NA	24 hr	97.5	22.3	7.494	35.6	25.5	7.445	
23063	73	Control	5.45	813	.45-caliber	Control	148	Control	810	6.2	Control	84.9	34.9	7.431	44.1	37.7	7.419	Cardiac and
											15 min	107.2	25	7.421	39.1	38.8	7.359	right and
		30 min	4.20			30 min	148	30 min	1020	120	30 min	82.2	35.5	7.433	45.5	35.3	7.381	left
											45 min	84.9	33.1	7.398	43.7	36.3	7.388	pulmonary
											60 min	87.3	34.4	7.421	45.5	35.6	7.390	contusions
		24 hr	NA			24 hr	NA	24 hr	NA	NA	24 hr	No sample			No sample			

Three animals (59, 60, and 61) had no morphologic pulmonary or cardiac injuries with projectile velocities of 840, 834, and 852 ft/sec.

One animal (57) had no evidence of epicardial or pulmonary damage. However, two cusps of the aortic valve were disrupted from the annulus of the aortic valve with surrounding subendocardial hemorrhage (figure 4). The projectile velocity was 834 ft/sec.

Figure 4. Disrupted Cusps of the Aortic Valve

One animal (63) was impacted with a .45-caliber projectile at 813 ft/sec. This animal died 10 hours post shot. At autopsy approximately 1.5 liters of blood was present in the right pleural cavity. There was a massive hemorrhage into the parenchyma of the right lung with a linear tear (5-cm) across the right hilum. A left lung contusion (approximately 150 cc) was present on the cardiac lobe. There was a superficial epicardial contusion across the posterior aspect of the right ventricle (figures 5 and 6).

In groups I and II, no projectile penetration of the armor occurred. All animals had a skin laceration as described in group I with the exception of the animal impacted by the .45-caliber bullet. This animal had a larger skin laceration (4- to 5-cm). No fractured ribs or pneumothoraxes occurred in this group.

1. Physiologic Monitoring.

a. Cardiac Output.

As in group I, no relation between the level of damage and cardiac output was observed. The animal impacted with the .45-caliber projectile did not complete the 24-hour

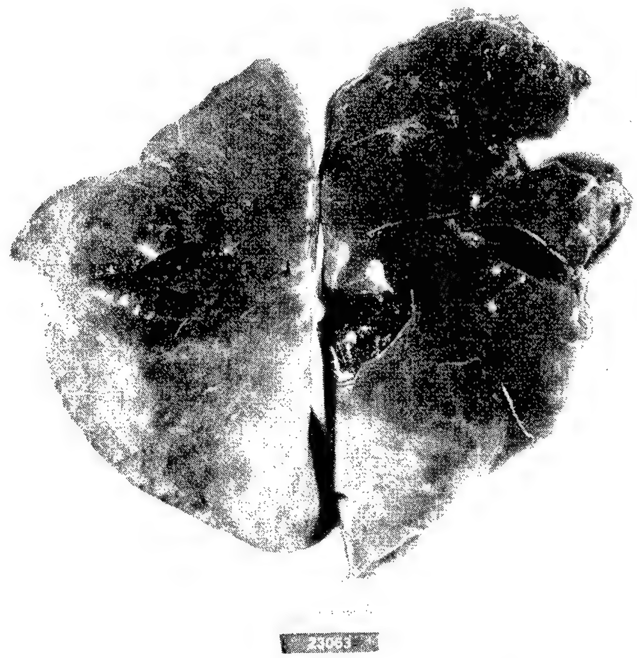

Figure 5. Linear Tear Across the Right Hilum.
Left Lung Contusion on Cardiac Lobe

Figure 6. Superficial Epicardial Contusion

protocol, expiring from wounds 10 hours post shot. However, at 30 minutes, no change in cardiac output, as compared to the preshot value, was observed.

b. EKG.

Nonspecific T wave changes were noted in most animals at 30 minutes and 24 hours. Again, no relation was noted between EKG changes and cardiac damage. No conduction defects or arrhythmias occurred in this group.

c. Serum CPK and LDH.

In the six animals impacted with the .38-caliber projectile, no relation between the level of cardiac and pulmonary damage and serum enzymes measured was observed. The .45-caliber shot resulted in marked elevation of measured serum enzymes at 30 minutes (see table 2).

d. Systolic Blood Pressure.

The systolic blood pressure was depressed in most animals during the second anesthesia at 24 hours. No relation between the level of damage and systolic blood pressure was observed. In the .45-caliber test, the systolic blood pressure at 30 minutes was unchanged from controls.

e. Blood Gas Data.

The one animal with aortic valve disruption had moderate hypoxemia at 24 hours.

All other animals in this group had normal 24-hour blood-gas measurements. Two of these animals had low control arterial and venous blood gases. The amimal impacted with the .45 caliber had normal blood gases at 30 minutes.

C. Group III.

The results for this group are summarized in table 3. As in groups I and II, small skin lacerations without rib fractures or pneumothoraxes were encountered in all nine animals. These animals were all impacted with .38-caliber projectiles at maximum diastolic filling of the heart. Figure 7 shows the typical record obtained when the firing of the projectile is synchronized with the electrical events of myocardial contractility.

Of the animals tested in this group none showed epicardial contusions nor injury to the great vessels or valvular structure of the heart.

Four animals (66, 69, 73, and 74) showed no evidence of right lung injury. The velocities of these shots were 820, 829, 821, and 829 ft/sec.

Four animals (64, 67, 70, and 71) had multiple right lung contusions usually involving the apical and diaphragmatic segments of the right lung. The velocities of these shots were 853, 830, 845, and 840 ft/sec.

One animal (65) had two solitary contusions of the right lung approximately 2 to 4 cc in volume. This animal was impacted with a .38-caliber projectile at 826 ft/sec.

Table 3. Summary of Results for Group III

Animal number	Weight	Cardiac output		Velocity	Projectile	Systolic BP		Enzymes			Arterial blood gas				Venous blood gas			Pathology
									LDH	CPK		pO_2	pCO_2	pH	pO_2	pCO_2	pH	
	kg	ℓ/min		ft/sec		mmHg			units			mmHg		units	mmHg		units	
23064	44.6	Control	3.76	853	.38-caliber	Control	112	Control	975	8	Control	106.2	34.9	7.398	63.2	31.5	7.379	Multiple right lung contusions
											15 min	89.7	35.2	7.407	51.4	36.0	7.378	
		30 min	4.48			30 min	104	30 min	950	19.2	30 min	89.3	32.6	7.387	47.4	33.3	7.389	
											45 min	89.8	34.8	7.382	49.1	33.1	7.370	
											60 min	80.5	34.0	7.371	39.4	35.0	7.396	
		24 hr	4.33			24 hr	104	24 hr	1250	74.5	24 hr	96.9	33.1	7.612	37.3	35.9	7.583	
23065	48	Control	4.36	826	.38-caliber	Control	136	Control	715	16	Control	94.4	33	7.414	55.8	33.1	7.352	Contusion two areas right lung
											15 min	95.3	32.2	7.431	58.0	32.0	7.403	
		30 min	3.89			30 min	108	30 min	785	33.6	30 min	103.0	30.7	7.441	59.1	32.6	7.398	
											45 min	105.5	31.4	7.433	59.8	36.0	7.402	
											60 min	103.9	31.9	7.419	41.2	33.7	7.397	
		24 hr	3.85			24 hr	88	24 hr	720	38.5	24 hr	96.1	32.9	7.505	38.4	36.0	7.483	
23066	55.8	Control	4.50	820	.38-caliber	Control	120	Control	545	9.4	Control	101.0	38.3	7.441	53.5	39	7.461	No pathology
											15 min	102.7	37.9	7.483	60	38.9	7.475	
		30 min	4.71			30 min	116	30 min	575	15.0	30 min	87.8	40.9	7.505	51.6	39.3	7.467	
											45 min	112.7	35.6	7.532	58.8	37.7	7.496	
											60 min	112.1	36.1	7.573	60.6	35.4	7.497	
		24 hr	4.22			24 hr	100	24 hr	830	32	24 hr	91.1	36.2	7.441	31.9	38.4	7.474	
23067	50.6	Control	7.70	830	.38-caliber	Control	132	Control	535	18.9	Control	82.5	40.1	7.421	59.7	38.6	7.402	Multiple right lung contusions
											15 min	99.7	37.8	7.453	64.5	31.6	7. 4	
		30 min	5.03			30 min	108	30 min	580	87.0	30 min	94.1	37.7	7.491	53.3	38.9	7.445	
											45 min	100.2	38.3	7.493	58.1	39.8	7.474	
											60 min	111.6	35.3	7.498	42.9	36.6	7.493	
		24 hr	4.60			24 hr	92	24 hr	860	44.0	24 hr	80.4	36.0	7.536	42.9	36.6	7.493	
23069	48	Control	3.99	829	.38-caliber	Control	124	Control	1275	27.5	Control	119.1	32.3	7.406	70.1	31.4	7.403	No damage
											15 min	125.7	29.4	7.398	68.7	31.8	7.448	
		30 min	4.40			30 min	132	30 min	1150	30.8	30 min	123.4	32.8	7.441	63.5	33.6	7.405	
											45 min	119.5	29.5	7.450	46.3	28.6	7.439	
											60 min	48.1	32.1	7.405	39.8	29.6	7.390	
		24 hr	1.93*			24 hr	92*	24 hr	950	30.5	24 hr	95.5	25.1	7.438	34.3	30.5	7.384	
23070	41.6	Control	4.11	845	.38-caliber	Control	108	Control	1215	20.8	Control	81.5	33.1	7.420	57.4	30.8	7.346	Right lung contusions
											15 min	73.3	37.1	7.359	51.9	38.6	7.341	
		30 min	2.96			30 min	94	30 min	1160	29.8	30 min	81.0	33.1	7.358	56.9	38.6	7.328	
											45 min	85.9	29.3	7.854	58.6	33.2	7.335	
											60 min	79.5	37.7	7.360	52.1	37.4	7.312	
		24 hr	3.18			24 hr	80	24 hr	1200	50.5	24 hr	87.8	28.1	7.384	42.8	35.8	7.306	
23071	77.4	Control	4.91	840	.38-caliber	Control	136	Control	800	6.4	Control	113	33.2	7.527	42.9	35.2	7.571	Right lung contusions
											15 min	116.5	29.1	7.655	37.6	31.7	7.631	
		30 min	5.44			30 min	144	30 min	845	9.2	30 min	115.5	27.6	7.678	32.6	32.0	7.728	
											45 min	113.8	24.8	7.738	34.1	36.2	7.641	
											60 min	106.8	33.2	7.658	32.5	43.7	7.517	
		24 hr	4.82			24 hr	96	24 hr	875	19.0	24 hr	93.0	25.8	7.648	37.4	30.0	7.520	
23073	49.5	Control	5.96	829	.38-caliber	Control	136	Control	NA	NA	Control	63.3	31.7	7.528	38.6	34.2	7.469	Minor right lung contusion
											15 min	62.6	34.2	7.498	35.1	37.5	7.407	
		30 min	4.60			30 min	96	30 min	NA	NA	30 min	88.8	32.6	7.580	33.8	33.9	7.534	
											45 min	108.9	30.3	7.598	28.0	38.8	7.455	
											60 min	95.8	33.7	7.588	30.8	39.8	7.506	
		24 hr	5.11			24 hr	88	24 hr	NA	NA	24 hr	92.6	23.7	7.479	39.1	24.7	7.460	
23074	48.5	Control	4.93	821	.38-caliber	Control	128	Control	NA	NA	Control	97.7	27.8	7.417	53.7	32.5	7.411	No damage
											15 min	103.5	29.1	7.447	47.2	31.4	7.428	
		30 min	3.49			30 min	116	30 min	NA	NA	30 min	96.2	34.6	7.458	45.1	32.6	7.407	
											45 min	104.6	27.2	7.457	48.9	32.5	7.391	
											60 min	98.6	32.2	7.421	–	–	7.392	
		24 hr	4.86			24 hr	112	24 hr	NA	NA	24 hr	100.8	19.9	7.549	39.4	23.9	7.510	

*Hemorrhage from displaced carotid catheter prior to 24-hour testing.

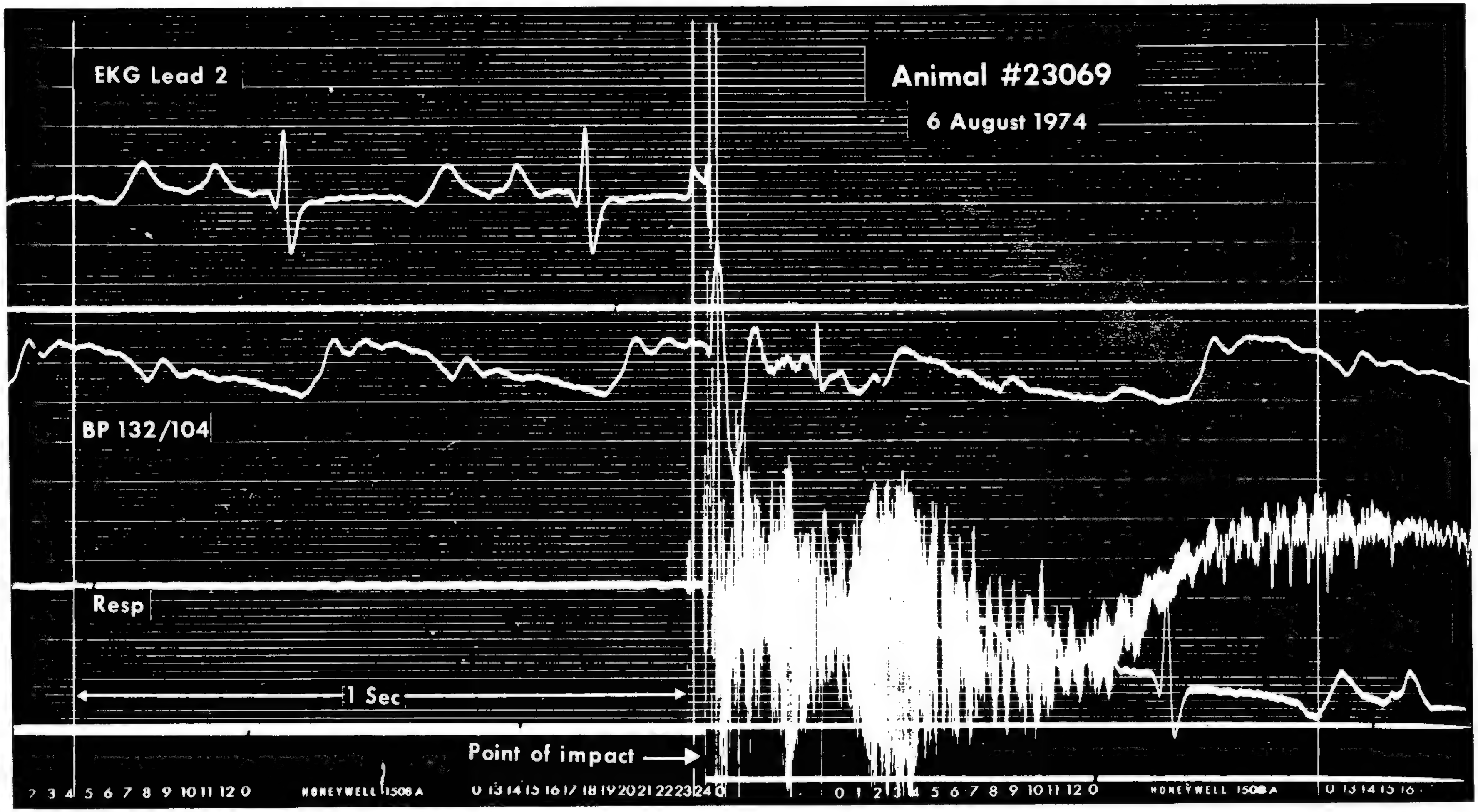

Figure 7. Typical Record for Synchronizing Projectile Firing with Electrical Events of Myocardial Contractility and Respiratory Cycle

1. Physiologic Monitoring.

a. Cardiac Output.

In the nine animals tested, no consistent relation was noted between post shot cardiac output at 30 minutes and 24 hours and the severity of right lung damage.

b. EKG.

Nonspecific T wave changes were again apparent in all animals. No conduction defects or arrhythmias were noted.

Our experimental procedure for producing blunt trauma on the heart and lungs differs markedly from the mechanism involved in automobile accidents. The automobile injury is characterized by a large-mass, low-velocity impact. In contrast, our experimental design utilizes a low-mass, high-velocity impact applied for an extremely short interval over a small body area. The effects produced by these two systems may not be comparable.

In the group-I animals, the amount of muscular cardiac damage produced was quite small. The function of the heart was not impaired. Two animals in this group did demonstrate serious aortic or valvular damage. This damage was initially believed to have been caused by the catheter used to monitor left-ventricular end-diastolic pressure. The catheter (figure 8) is rather stiff and the injuries to the aorta and aortic valve could have been relatively easy to produce. More difficult to explain were the multiple contusions, of the right lung only, which occurred in animals 54 and 56. Both impacts were on the left chest.

Figure 8. Left-Ventricular End-Diastolic Catheter in Place

The end diastolic catheter was not used in group II. One animal (57) in this group experienced disruption of two of the three leaflets of the aortic valve. While velocities of the .38-caliber projectile in this group were as high as 852 ft/sec, no right lung contusions were produced.

c. Serum CPK and LDH.

Here again, the degree of enzyme rise could not be related to the extent of underlying right lung damage.

d. Systolic Blood Pressure.

As noted in groups I and II, the systolic blood pressure measured at 24 hours post shot was depressed in most animals. Again, no consistent relation was found in this group between the level of blood pressure and the underlying pulmonary injury.

e. Blood Gas Data.

Two of the animals (66 and 69) with no gross pulmonary contusions demonstrated mild desaturation of arterial O_2 at 24 hours.

Two of the animals (64 and 71) with right lung contusions demonstrated mild desaturation of arterial O_2 at 24 hours.

No consistent trends in arterial or venous blood gas determinations were noted in the nine animals tested.

IV. DISCUSSION.

From previously published reports by DeMuth and others,[2] myocardial contusion accounts for a substantial number of mortalities involving blunt trauma to the chest in automobile accidents. In most of these circumstances, the blunt trauma was produced by steering-wheel impact. These groups found a good correlation between the electrocardiographic events after the injury and the extent of myocardial damage.

In the .45-caliber test, severe right lung damage with laceration through the pulmonary hilum was produced. This was the cause of the animal's death. The 234-grain, .45-caliber projectile at a velocity of 800 ft/sec has a striking kinetic energy 48% above the 158-grain, .38-caliber projectile at a similar velocity.

In group III, with impact over the open "cardiac window," i.e., over the point of maximal cardiac impulse with maximal diastolic filling during expiration, right lung damage could be consistently produced. These reproducible injuries were noted when the impact velocity was greater than 830 ft/sec. One animal (65) experienced two small right lung contusions when the impact velocity was 826 ft/sec.

[2]DeMuth, W. E., and Finsser, H. F. Myocardial Contusion. Arch. Intern. Med. *115*, 434-442 (1965).

Similar results, contralateral lung injury, have been described in blast-induced injury.[3] The most likely explanation involves the concept of a hydraulic system interposed between the impact site and the right lung. With a pure pulmonary impact site without an intervening heart no contracoup lung injury is produced. In this system, the impacted lung acts as a pneumatic system, easily compressed and absorbing the transmitted force rather than transmitting it to the right lung. When a filled heart is interposed between the impact site and the right lung (left lung retracts with expiration), the impact force is transmitted through the blood (fluid) media. This occurs because fluid is much less compressible than air, therefore, less force is absorbed. This force is transmitted to the right lung, producing pulmonary contusion; thus, a hydraulic effect is created by a filled heart.

The absence of significant EKG changes even in animals with gross cardiac damage would seem to indicate that the goat heart muscle tolerates the injuries involved in our study quite well without severe alteration in physiologic performance. This seems evident in the lack of severe impairment of cardiac output and systolic blood pressure alterations.

While multiple lung contusions were produced in four Group-III test animals out of a total of nine impacted with the .38-caliber projectile, blood gas changes did not permit differentiation between these animals and those having no pulmonary damage. Perhaps these multiple pulmonary contusions do alter pulmonary function greatly. The design of the protocol permitted survival for only 24 hours post shot. It is possible these lesions may contribute to physiologic derangement after 24 hours producing a "posttraumatic pulmonary insufficiency." The natural history of these morphologic pulmonary lesions has not been defined.

The absence of consistent relationships between response variables and the severity of the underlying trauma has been noted several times in this paper. The question is further explored in the appendix.

V. CONCLUSIONS.

1. In the goat, the myocardium is relatively resistant to the blunt trauma produced beneath 7-ply Kevlar body armor when impacted with a .38-caliber projectile at velocities less than 830 ft/sec.

2. In the goat, protected by 7-ply Kevlar armor, valvular and possibly great vessel damage can be produced when impacted with a .38-caliber projectile at 800 ft/sec.

3. In the goat protected by 7-ply Kevlar armor, multiple right lung contusions can be produced when the animal is impacted on the left chest during simultaneous diastole and expiration by a .38-caliber projectile at 830 ft/sec.

4. In the goat protected by 7-ply Kevlar armor, the .45-caliber projectile at 800 ft/sec produces serious intrathoracic damage despite non penetration of the projectile.

5. In a clinical setting, patients wearing the 7-ply Kevlar body armor impacted over the chest should be hospitalized for observation despite normal chest X-ray, electrocardiogram, and arterial blood gases.

[3] White, Clayton S., M.D., and Richmond, Donald R., Ph.D. Blast Biology. *In*: Clinical Cardiopulmonary Physiology. R. C. Kory and B. L. Gordon, Editors. Chapter 63, pp 974-992. Grune and Stratton, Inc., New York, New York. 1960.

VI. SUMMARY.

Twenty-three Texas Angora goats underwent left chest impacts with either the .38-caliber or .45-caliber projectile while protected with 7-ply Kevlar-29 body armor (400/2 denier). In the 22 animals shot with the .38-caliber bullet, 2 had small physiologically nonsignificant myocardial contusions, 1 had aortic valvular disruption, 7 had contralateral right pulmonary contusions occurring with projectile velocities of 836 ft/sec or greater.

In the animal shot with the .45-caliber projectile massive contralateral, right lung damage was produced.

A mechanism for the production of contralateral right pulmonary contusions is discussed.

APPENDIX

RESPONSES TO SEVERITY OF INJURY

The following procedure was used to see whether the observed changes in some of the response variables (cardiac output, systolic blood pressure, etc.) were related to severity of injury.

First, the pathologies of the animals were ranked in order of increasing severity as follows:

Rank	Pathologies	Animals
1	No damage	51, 59, 60, 61, 66, 69, 74
2	Small contusions, cardiac lobe left lung	50, 53, 58, 62
	Two contusions, right lung, about 2 to 4 cc	65
	Minor right lung contusions	73
3	Multiple right lung contusions	56, 64, 67, 70, 71
4	Aortic lacerations and multiple right lung contusions	54
5	Heart contusion and small pulmonary contusion	52
	Small heart contusion and torn aortic valve cusp	55
	Two cusps of aortic valve disrupted from annulus and subendocardial hemorrhage	57
6	Heart contusion and right lung contusion with massive hemorrhage and left lung contusion, about 155 cc	63

For the response variable cardiac output, a score was computed as follows: For each animal, the maximum overall control and postshot values were divided by the minimum overall control and postshot values, and the natural logarithm of the ratio taken. Each of the numbers so computed was then divided by the mean of these numbers, over all the animals, to give each animal a derangement score for cardiac output. Scores for systolic blood pressure and for the enzymes LDH and CPK were computed in the same way. These scores reflect the fluctuation of a given variable in a given animal, as compared to the mean fluctuation of that variable over the whole

group. Large fluctuations give high scores and small fluctuations give low scores. For ApO_2, $ApCO_2$, VpO_2, $VpCO_2$, ApH, and VpH (where control values were not available for some animals), the maxima and minima were taken over postshot values. For ApH and VpH, (maximum – minimum) was computed rather than ℓn (maximum/minimum).

For each of these 10 response variables, a point plot was made of derangement score versus Pathology Severity Rank (figures A-1 through A-10). On none of these point plots was there visible a consistent pattern of high scores in more severely injured animals and low scores in the less severely injured, or vice versa.

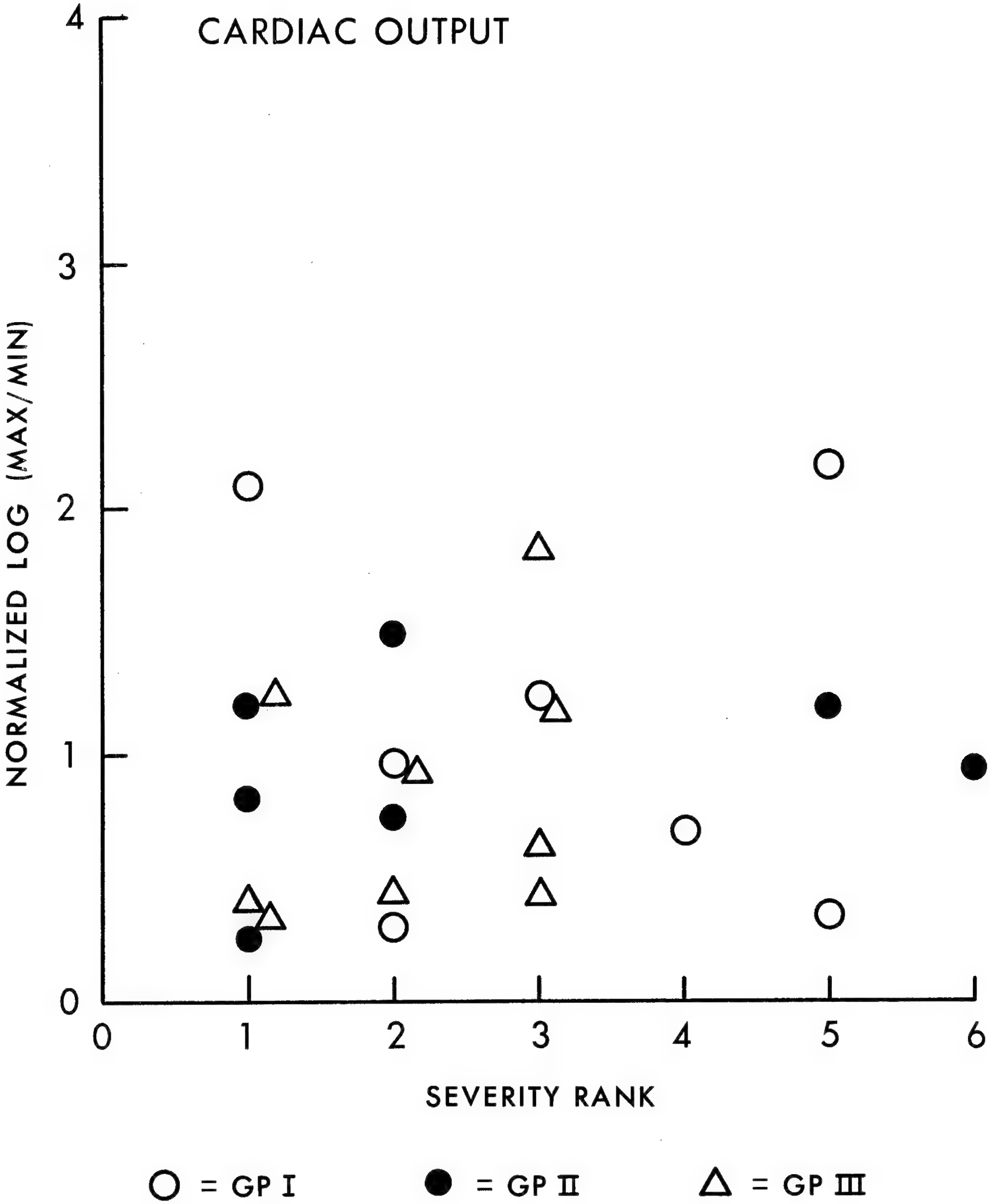

Figure A-1. Cardiac Output Versus Severity of Injury

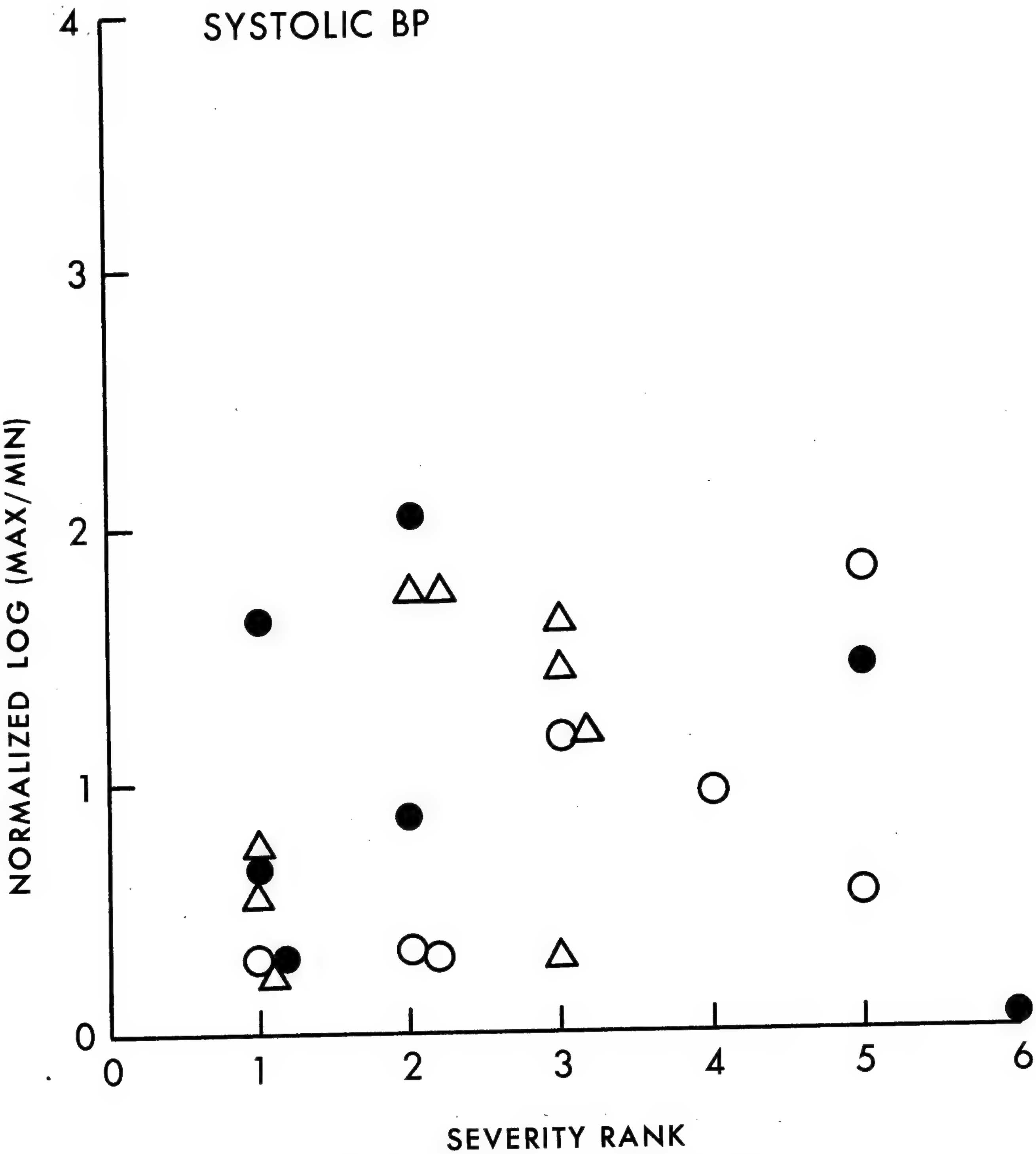

Figure A-2. Systolic BP Versus Severity of Injury

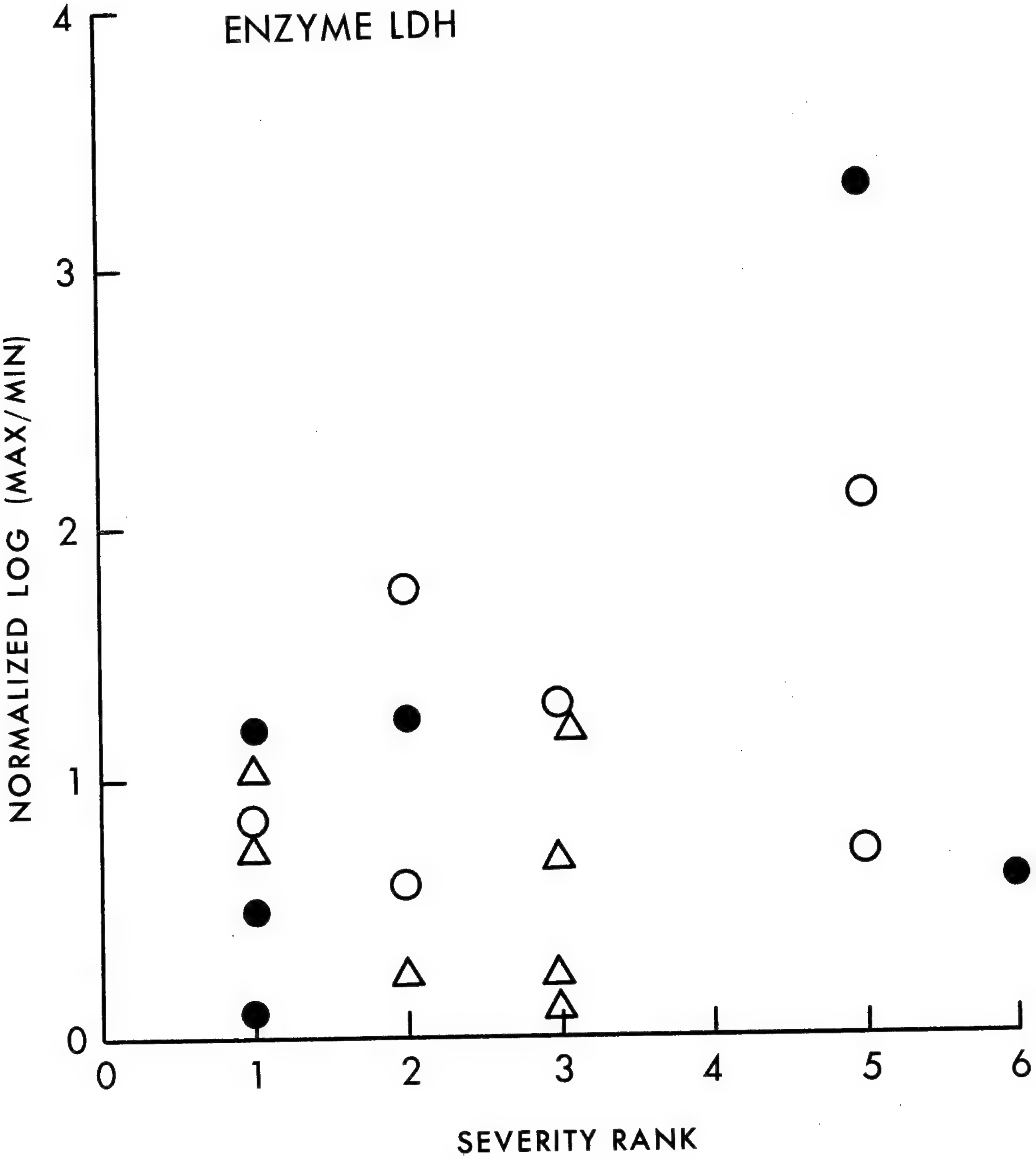

Figure A-3. Enzyme LDH Response to Severity of Injury

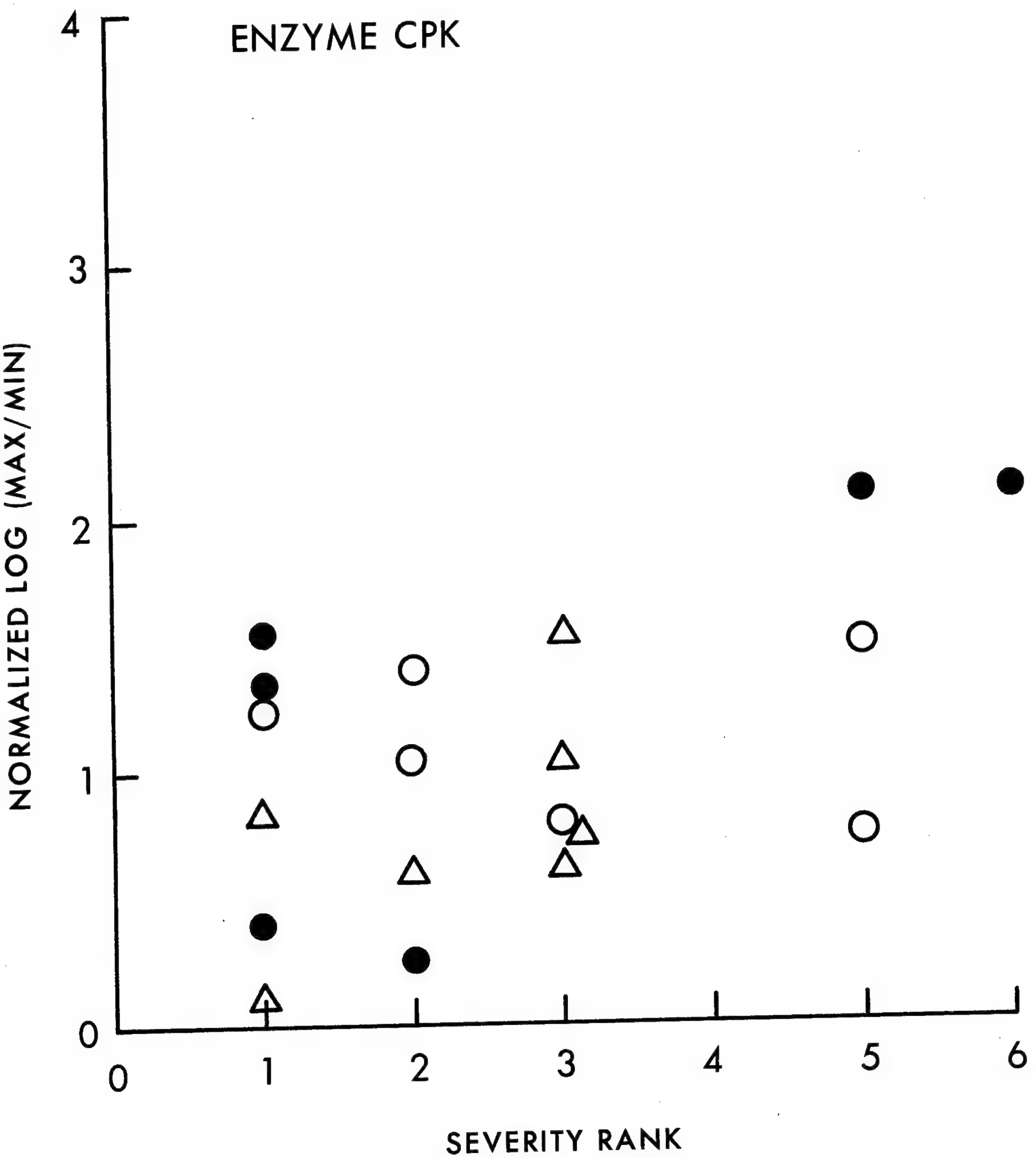

Figure A-4. Enzyme CPK Response to Severity of Injury

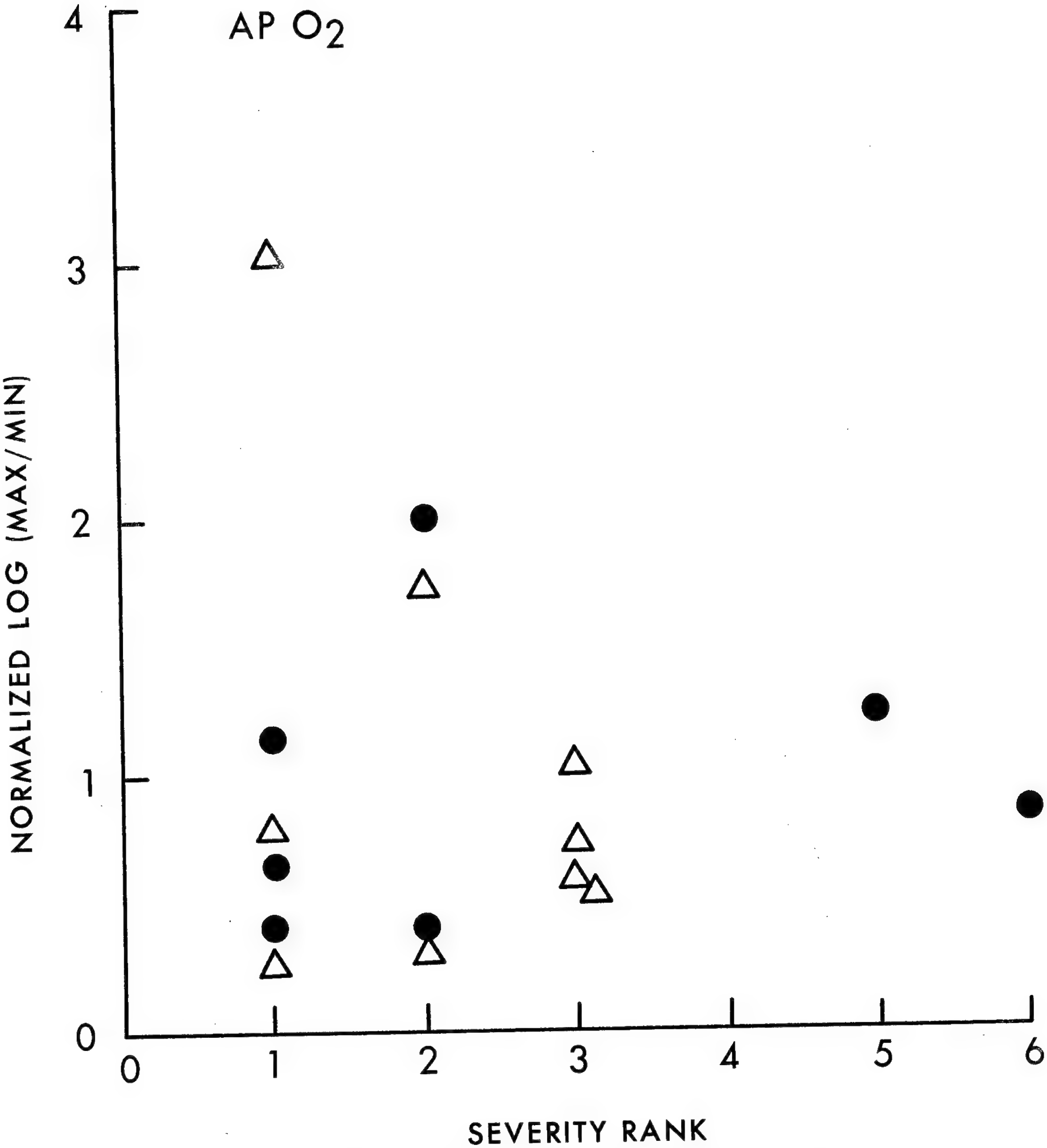

Figure A-5. Arterial Pressure of Oxygen Versus Severity of Injury

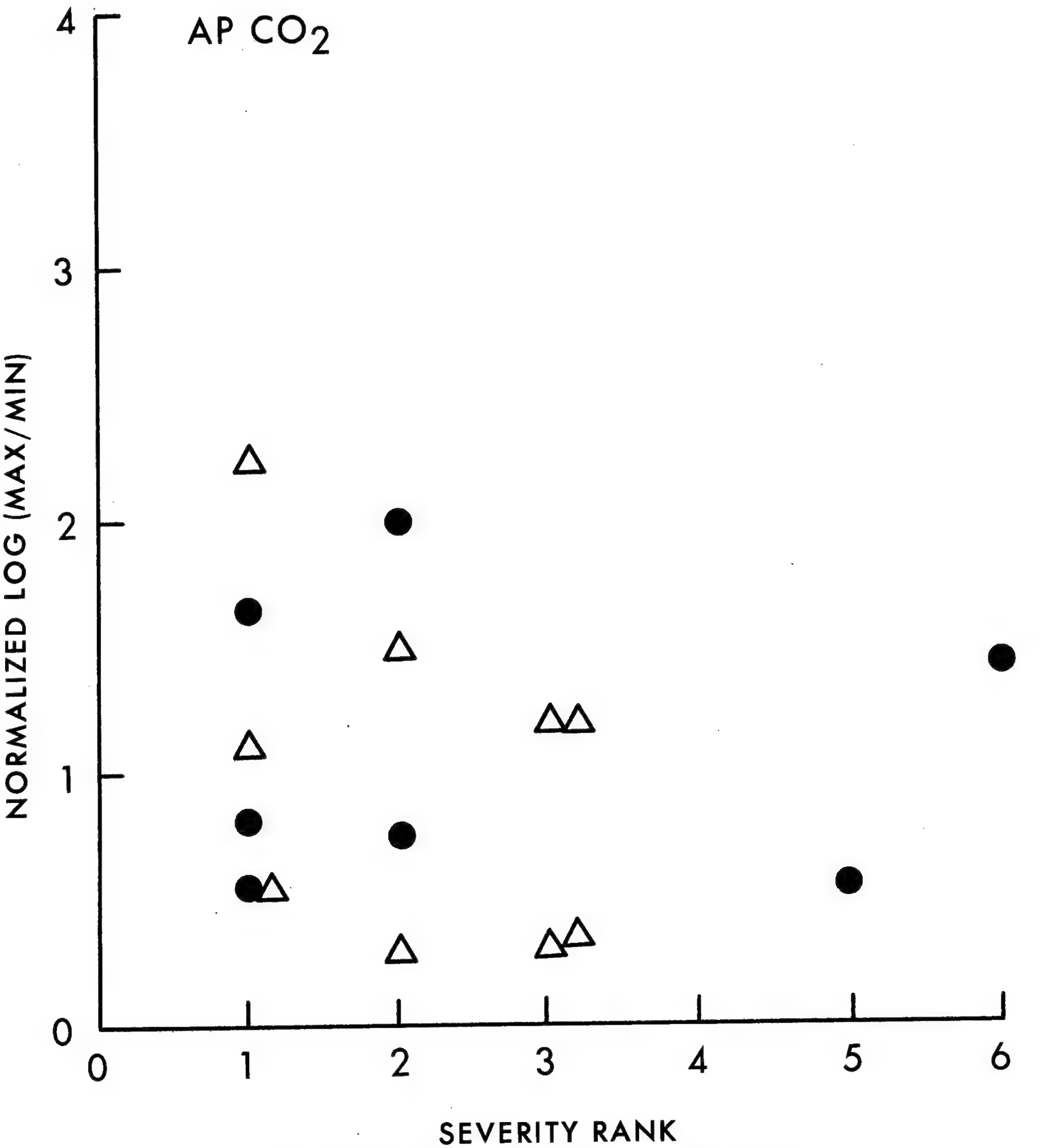

Figure A-6. Arterial Pressure of CO_2 Versus Severity of Injury

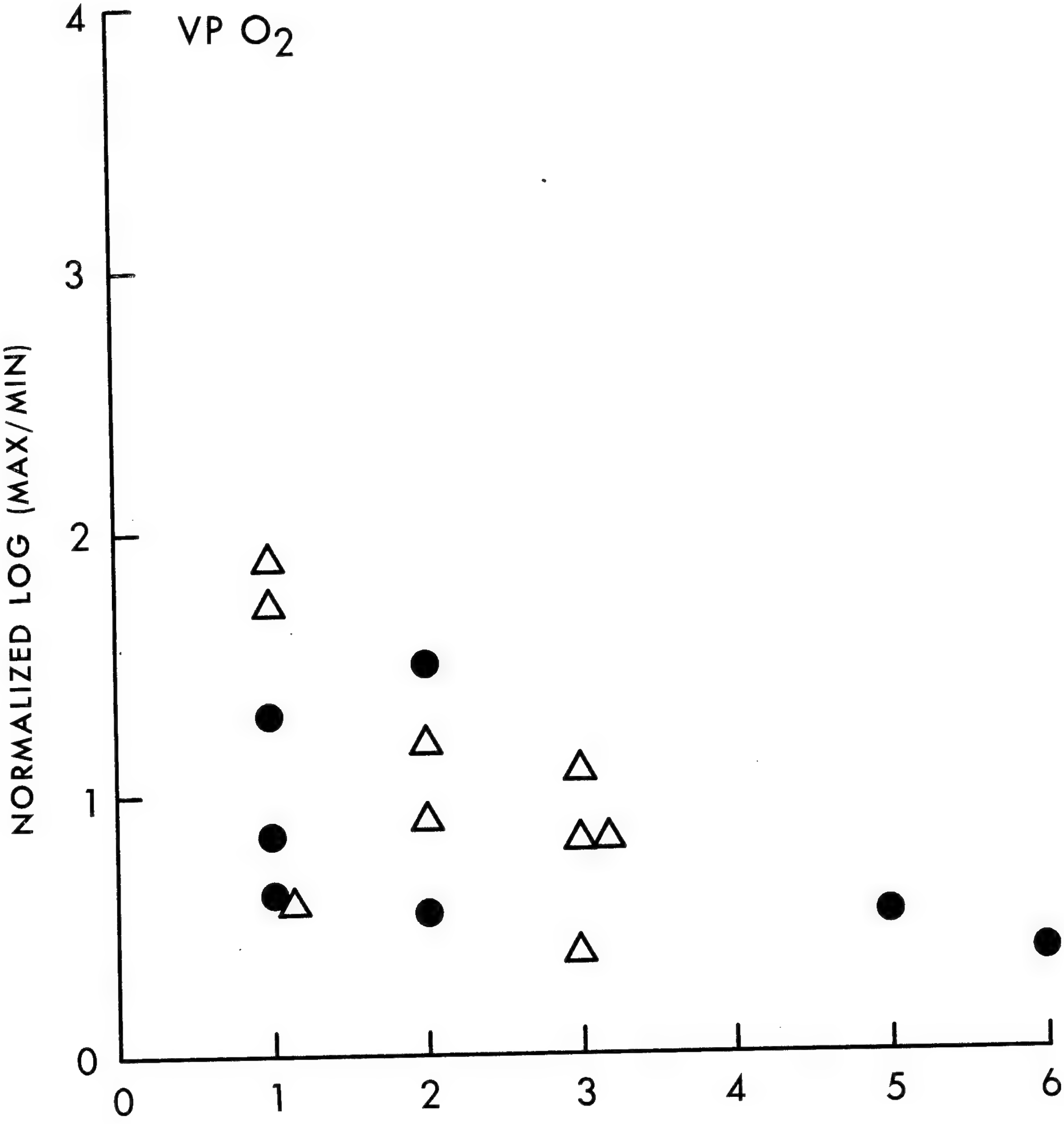

Figure A-7. Venous Pressure of Oxygen Versus Severity of Injury

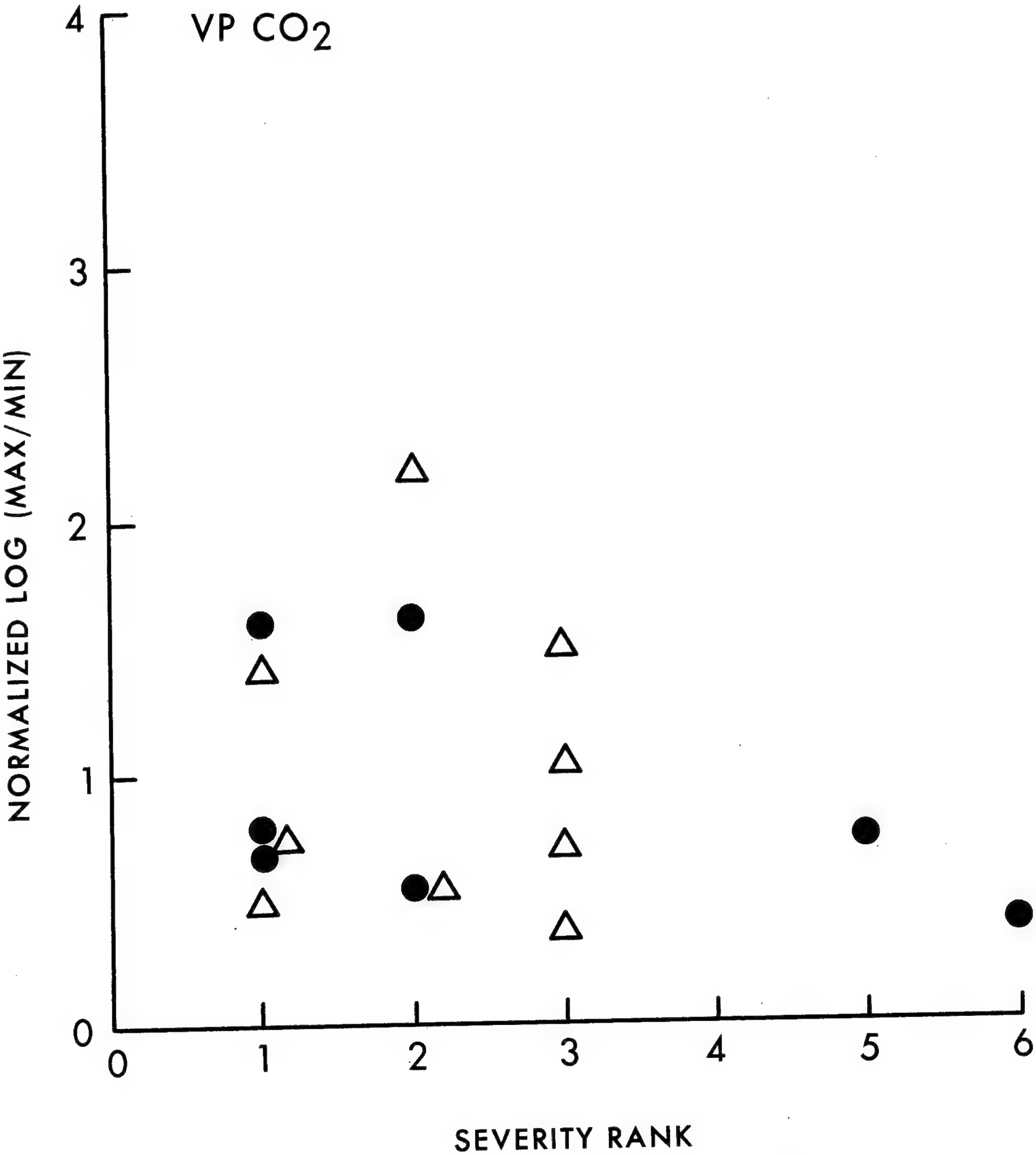

Figure A-8. Venous Pressure of CO_2 Versus Severity of Injury

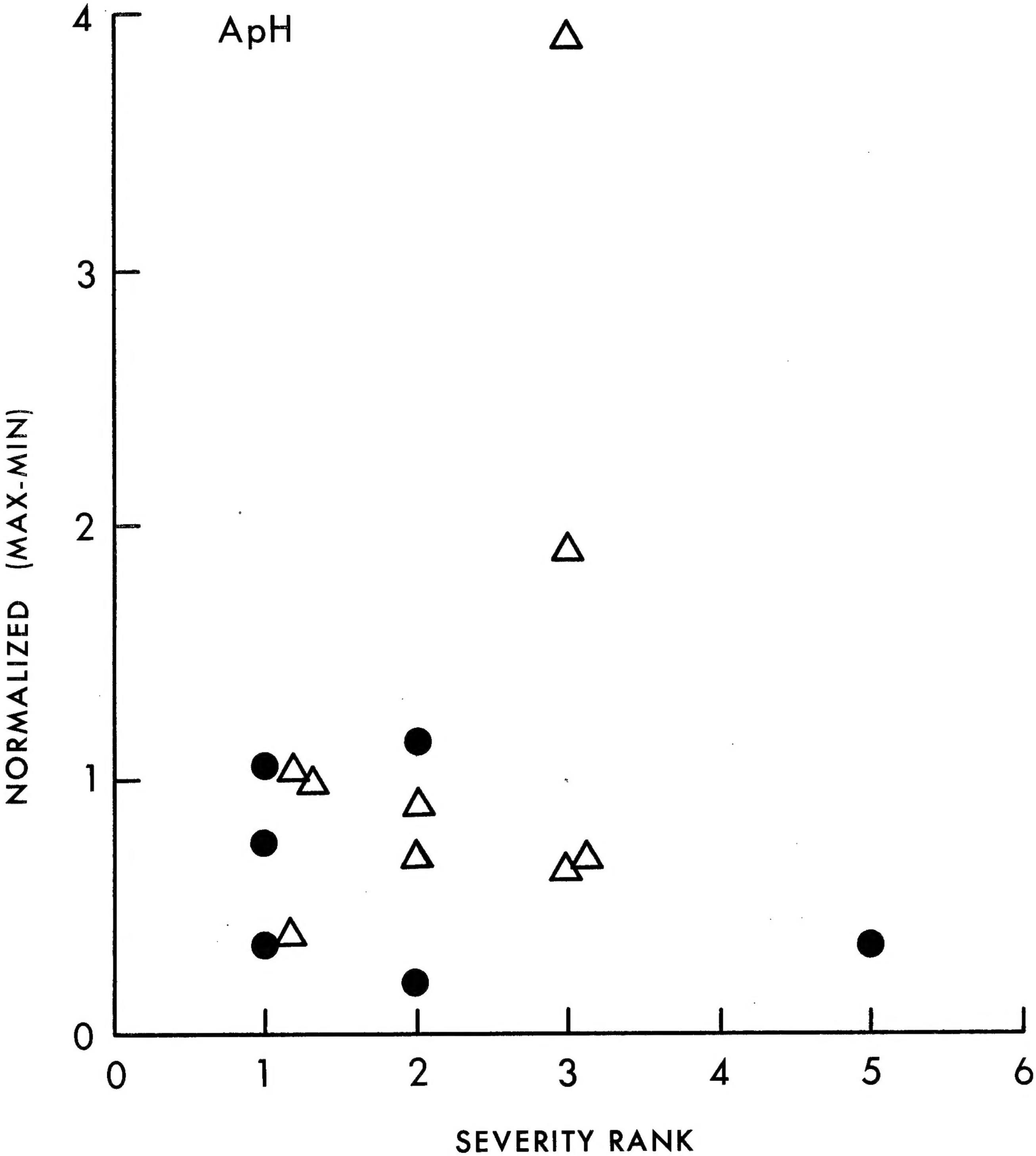

Figure A-9. Arterial pH Versus Severity of Injury

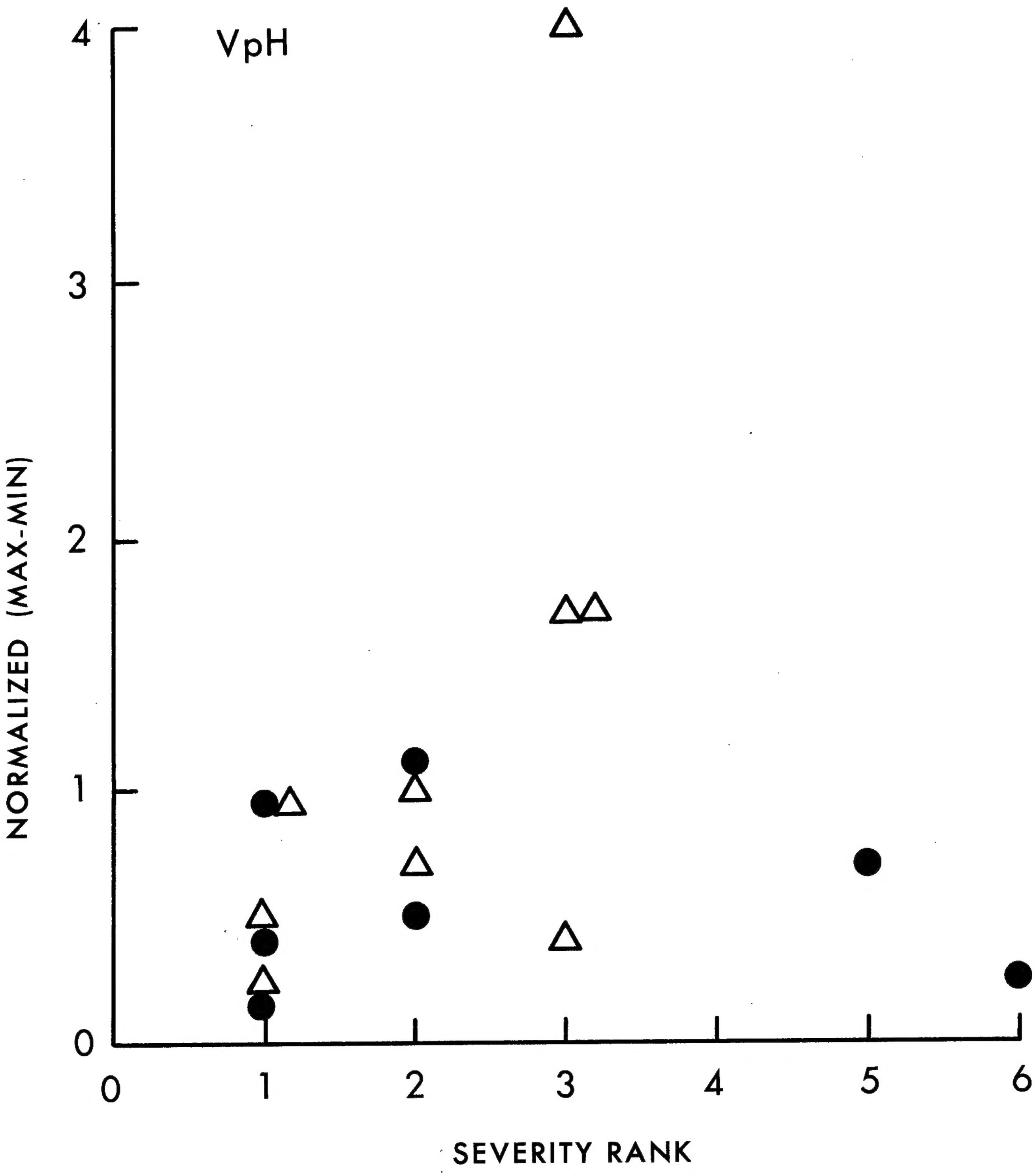

Figure A-10. Venous pH Versus Severity of Injury

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